Lumbo-sacral adhesive arachnoiditis: a review

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'Lumbar arachnoiditis easily qualifies as a medical "purple cow" with few surgeons ever having seen it and fewer wishing to. Despite desire, lumbar arachnoiditis represents a serious complication of inflammation, trauma, and pia-arachnoid foreign body reaction'[1].

Introduction

Lumbo-sacral adhesive arachnoiditis (LSAA) is a condition of fibrous invasion of the pia mater of the cauda equina. The fibrosing process may be secondary to deformity of the spinal column, to the sequelae of physical injury to the spine, or to toxic effects of introduced noxious substances.[2] The symptoms of LSAA are bizarre, and commonly dismissed as neurotic or functional, if not confused with sciatic and rheumatic syndromes.

This paper is not primarily intended for the attention of radiologists, neurologists, and neurosurgeons who are aware of the prevalence of post-myelography arachnoiditis; it is directed at other specialists such as rheumatologists and general practitioners who might consider the re-diagnosis of patients with seemingly bizarre symptoms and signs. They might inform the Committee for Safety in Medicine[3], and direct patients to a Self Help Group. The Arachnoiditis Self-Help Group in Great Britain has listed 350 members since its formation in 1988 (see end of references for address).

There may be many thousands of patients in Britain and all over the world who are ignorant of the cause of their suffering and who may not be aware that a mutually supportive Self Help Group exists. It is also hoped that the problem of this distressing iatrogenic disease might be brought to public conscience, so that sympathy and understanding be given to sometimes embittered and often lonely sufferers.

In spite of the fact that the oil-based iodine agents have been abandoned there is still the risk that water-based iodine preparations will in some cases cause arachnoiditis[4]. The present trend towards the use of non-invasive electronic scanning techniques brings hope that myelography using iodine-based media or indeed any other medium will become a thing of the past.

Aetiology of arachnoiditis

Table 1 shows the aetiological factors producing lumbar arachnoiditis in the series of 38 patients described by Quiles, Marchiello, and Tsairis at the Hospital for Special Surgery, New York and the New York Hospital[5]. Injection of contrast media and operations for discectomy and laminectomy account for most of the occurrences of lumbar arachnoiditis.

Table 1. Aetiological factors producing Lumbar Arachnoiditis in 38 patients studied

Type of insult	*No. of patients*
Agents injected into subarachnoid space	
Contrast media	36
Anaesthetic agents	2
Intradural steroids	1
Infection of intervertebral space	1
Trauma vertebral and paravertebral injuries	
Back manipulation	1
Fracture lumbar spine	1
Removal of fracture of coccyx	1
Removal of bone splinters from spinal canal	1
Spinal operations	
Extradural	
Discectomy	29
Laminectomy and exploration	22
Lumbar spine fusion	11
Cervical laminectomy	1
Anterior cervical fusion	2
Cervical epidural haematoma	1
Intradural	
Rhizotomy	3
Closure of spinal fistula	2
Foreign bodies of previous dural sutures	2
Durotomy	1
Nerve root severed	1
Space occupying lesions	
Neurofibroma	1
Intrathecal haemorrhage	
Bloody tap	2
Miscellaneous	
Pseudospondylolisthesis	1
Radiation of lumbar area for lymphosarcoma	1
No previous surgery or known trauma	2

Pathology of arachnoiditis

The pia mater is very fragile and sensitive to both chemical and physical injury. The response to injury of whatever nature is similar to that after injury to peritoneum, pericardium, and pleura. Vascular damage in the dural and peridural tissues prevents normal hypervascularization. The absence of hypervascularization yields few enzyme bearing leucocytes. Phagocytes and fibrolytic enzymes are probably washed away or diluted by the cerebrospinal fluid, thereby rendering the arachnoid incapable of eradicating these fibrous bands. The fibrinous bands are invaded by fibrocytes which lay down collagen. Calcification of the fibrous bands may occur[5]. There is invasion of the arachnoid fibrils by collagen cells, with adhesions causing enmeshment and constriction of constituents of the cauda equina. Sections of the veins are constricted by scar tissue and this leads to engorgement of the distal parts of the veins. Globules of the oily iodine medium are often enmeshed in the dense scar tissue. The process of enmeshment is insidious, and may not cause symptoms for many

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Table 2. Gross pathological findings in 22 patients who underwent surgery for segmental and diffuse arachnoiditis[5]

	No. of patients
No dural pulsations	19
Meningeal thickening	20
No cerebrospinal fluid	17
Foreign body reaction	2
Fibrinoid adhesions	5
Oedema of the nerve roots	2
Nerve roots embedded in thick fibrous tissues	13
Cystification of Pantopaque	3
Bone or calcified plaques within dural tube	7

Table 3. Clinical manifestations of lumbar arachnoiditis in 38 patients studied[6]

	No. of patients
Symptoms	
Back pain	29
Leg symptoms	24
Back pain and leg symptoms	24
Signs	
Postural and mechanical findings	
List	1
Scoliosis	1
Increased lumbar lordosis	4
Loss of lumbar lordosis	1
Limited straight leg raising <70	16
Back tenderness	7
Limited trunk mobility	9
Muscular involvement	
Paraspinal muscular spasm or contracture	4
Atrophy	12
Neurologic involvement	
Anaesthesia or hypaesthesia	8
Hyporeflexia	
Knee jerks	5
Ankle jerks	25
Weakness	14
Urinary sphincter dysfunction	10

years. When the arachnoiditis is associated with anatomical deformity of the spine, the symptoms originate at the level of the part of the spinal column which is involved. Jayson[6] has shown that there is an enzyme defect in the blood of arachnoiditis patients, the correction of which is being studied with hopes of modifying the harmful effects of this disease.

Table 2 lists the gross pathological findings in a series of 38 cases analysed by Quiles *et al.*[5] In the face of such severe damage to most sensitive tissues, the therapeutic urge must be inhibited.

Symptoms of arachnoiditis

No two cases are alike either in the time-lapse before symptoms appear or in the distribution of the syndrome pattern. There are, however, certain bizarre symptoms which serve as clues to indicate the presence of arachnoiditis. The patient will describe 'burning sensation at the sacral area', gripping or clawing pains in the calves and ankles, severe pain down the back of one or both legs not of sciatic distribution, tingling pain in the insteps, burning at the inner aspects of the knees, tingling and pain in the feet. The symptoms often persist at rest and at night. Some patients need a bed cradle to protect the feet and ankles. Many need crutches and electric scooters, while others are bedridden. Table 3 lists the clinical manifestations of lumbar arachnoiditis in 38 patients studied by Quiles *et al.*[5]

Arachnoiditis due to aqueous non-ionic iodine solutions, and other media

Hurst[7] showed in 1955 that intrathecal injection of nonionic detergents produced LSSA. Cheung *et al.* described in vitro testing for the risk of arachnoiditis from aqueous contrast media[8]. The therapeutic local administration of Deomedrol and other steroids have caused LSAA. Lumbar arachnoiditis was described by Kaplan *et al.*[9] in a patient who had been submitted to myelography using thorium trioxide 15 years previously.

Postoperative adhesive arachnoiditis

Failure of laminectomy to relieve low back syndromes is relatively common[10]. Retained surgical swab debris is a causative factor in post laminectomy arachnoiditis[11]. Free blood and foreign bodies such as missiles may cause LSAA. Institutions involved in the rehabilitation of 'failed back surgery syndrome' patients have provided data that in about 11% of such patients the primary disease process producing incapacitation is LSAA. About 25% of the estimated 300 000 patients undergoing back surgery in the United States each year are not improved or made worse by surgery[10].

History of the use of iodine in oil or water as contrast media

Iophendylate was first used in 1944 and until the 1980s was being used for 350 000-450 000 myelograms per year. In Sweden iodophendylate was banned in 1948 when its role in the production of LSSA was identified. The toxicity of Iophendylate was confirmed by a comprehensive Symposium and published in *Spine* in March 1978[12]. In spite of the manufacturer's injunction to remove the opaque medium immediately after diagnostic X-ray, the oily substance was left in situ in many cases, and sometimes removed only after many months or years.

Water soluble agents containing ionic iodine are capable of producing arachnoiditis. The aqueous contrast medium metrizamide is a non-ionic solution of iodine safer than most other materials, but not completely free of risk. The profession should not be lulled into a sense of false confidence by claims of non-toxicity, and evidence of arachnoiditis must be sought for by follow-up of patients for many years. This applies also to metrizamide which is relatively safe. Follow-up is more easily said than done, since many years may elapse between the causative incident and the appearance of bizarre symptoms.

The nature of pain in LSAA

Arachnoiditis may occur without pain just as post-surgical intrathecal adhesions are usually painless. However, it is accepted that nerve fibres encased in collagenous scar tissue suffer an increase in neural tension, impairment in axioplasmic flow and neuro-humoral transport, with restriction of arterial supply and venous return. It has been shown by studying the

tail-flick test in mice that metrizamide causes significantly decreased tail-flick latencies, showing decreased pain thresholds[13]. The abnormal nerve fibres, unlike normal intraspinal nerve fibres, are capable of producing nociceptive discharge. The distribution of the lesions is so haphazard and specific to each individual case, that it is to be expected that no two cases have the same distribution and location of pain. However, the nature of the intractable pains from the lesions in the cauda equina is not only bizarre but specific and of diagnostic importance. Typically it is of a constant and burning nature suggesting an element of causalgia. It is a poorly localized paleospinothalamic pain pattern which is diffuse rather than spinothalamic pain which is sharp and well localized. Because of its constancy LSAA pain is more depressing and debilitating to patients, compared to more intense pain which is shortlived or subject to remission[2]. LSAA pain does not respond well to analgesics, and the patient usually needs to restrict normal activities, and to spend more time lying flat in bed. Careful elucidation of the exact nature and sites of pain is essential, because no other disease causes constant burning pain at the insteps, the inner aspects of the knees, and in the lumbosacroiliac area.

Clinical manifestations of lumbar arachnoiditis

Quiles *et al.*[5] analysed the clinical manifestations of lumbar arachnoiditis in 38 patients, as set out in Table 2. It is not surprising that many of these patients are treated for sciatica or other common painful diseases of the legs.

Case histories

The following case histories illustrate the progression from normal health or tolerable disability to tragic chronic invalidism which is suffered by a great number of patients throughout the world following invasive diagnostic or therapeutic trauma, and the need for re-assessment of the ethical justification for the use of noxious substances for so many years in spite of clear warning of the dangers involved. The legal problems are daunting, but should nevertheless be confronted.

Case 1

In November 1977 a 54-year-old woman complained of pain deep in the abdomen radiating to the spine at the level of the first lumbar vertebra. She was referred to a London teaching hospital where a myelography to exclude neoplasm showed no disease of the spine. The patient complained of a taste of iodine postoperatively. Three weeks later an exploratory operation to exclude retroperitoneal fibrosis revealed a pint of blood in the peritoneal cavity with endometriosis. Shortly afterwards the patient developed a burning sensation at the lumbosacral region and 'sciatic' pain in the right leg and tingling in the toes. After a delay of 2 years she was admitted to a neurosurgical unit for removal of the Myodil from the lower region of the spine. This minimized the sacral pain for some years, and she was able to enjoy a normal existence. The patient was not warned that the pain could recur in a severe form. In June 1979 she had a London Hospital/Imperial College left hip replacement, and in November 1979 a right hip replacement. The latter became loose and was replaced with a Charnley prosthesis in March 1980. In 1987 the patient developed pain in the right mid-thigh on walking or standing on the right leg due to loosening of the shaft of the prosthesis. Operation was delayed because there were confusing signs of neurological deficit due to arachnoiditis, associated with intolerable burning pains at the sacrum and at the inner aspects of the knees and at the insteps. After a successful fourth hip replacement in May 1988 the symptoms of arachnoiditis became worse. At present the patient is able to walk short distances with the aid of elbow crutches, and pain tablets. The pain is less when she lies flat, and this relief has been explained by assuming that the venous sinuses in the cauda equina become engorged in the standing position and are emptied when the patient lies flat. Nevertheless, the pain is less if the patient refrains from walking. An electric scooter allows her to accompany friends on walks and allows visits to the village shop. She has acquired an estate car which has no lip at the rear edge of the floor; with the aid of aluminium ramps it is proposed to load and transport the scooter to holiday venues.

Case 2

Dr EB, a female general practitioner aged 36 years was investigated for pain (?disc) in the right leg in 1954. Plain X-ray showed no abnormality. Treatment by manipulation did not help. A Myodil X-ray was performed. Tiny 'sacro-anal' cysts were seen, and most of the sacrum was removed. The Myodil was removed the following day. Postoperative symptoms included constant pain in the left leg. She was advised by The Mayo Clinic to avoid further operative treatment. She still has feelings that she is 'strung-up and sitting on a tight-rope and that two crabs are gripping my buttocks, and there is constant tingling in my legs.' (These symptoms are frequently experienced by other patients with arachnoditis.)

Case 3

A registered mental nurse (male) aged 48 years was investigated by myelography in April 1982. Several attempts were made to remove the oily substance immediately after the examination because he had a declared iodine allergy. He suffered from muscle spasms from the lower back to the calves.

In November 1982 fusion of the C5/6 vertebrae was performed, but the symptoms persisted. He retired due to ill health in February 1987. He can now walk for half-a-mile, but cannot stand still for more than a few minutes on account of pain and tenderness in his feet. He was referred to a London teaching hospital in 1983 where a further myelogram with a water-soluble contrast agent confirmed the diagnosis of arachnoiditis. Various drugs, anti-inflammatory, Tegretol, benzodiazipine, and tricyclic caused unacceptable side effects. Transcutaneous nerve stimulation and a partial lumbar sympathetic block failed to give relief. It is of interest that a medical tribunal failed to find any neurological abnormalities in the lower limbs, but agreed with the consulting physicians at the teaching hospital that the symptoms arose from arachnoiditis.

Safe alternative methods of diagnosing LSAA

High resolution enhanced computerised axial tomography (CAT) and non-enhanced magnetic resonance imaging (MRI) are capable of diagnosing LSAA.

Removal of oily and watery iodine contrast media

The manufacturers' instructions clearly advised that the Myodil should be removed at once after the X-ray examination. This involved a second lumbar puncture, and some surgeons and radiologists were reluctant to submit the patient to this manoeuvre. However, most authorities agree that when an opaque medium such as Myodil is found to be present on X-ray of the lower spine it should be removed. There have been reports of arachnoiditis occurring even after immediate removal of the opaque medium, since in some cases the small amount of iodine absorbed by the pia mater is sufficient to cause LSAA.

There have been research projects to study the occurrence of arachnoiditis after myelography and

laminectomy in experimental animals. Johansen *et al.*[15] showed that in the short term myelography did not cause arachnoiditis. This was misleading, since LSAA may develop many years after the insertion and indeed even after immediate removal of the noxious radio-opaque substance. On the other hand Cheung *et al.*[8] using in vitro testing for the risk of arachnoiditis from myelographic contrast media showed that ionic aqueous contrast media can cause LSAA, but Skalpe[16] states that non-ionic media such as metrizamide have not been found to cause arachnoiditis. Garancis and Haughton[4] showed that non-ionic water soluble metrizamide and meglumine iocarmate 'cause direct cellular injury in the arachnoid in the long term'.

Treatment of arachnoiditis

There is no cure for arachnoiditis, and no hope of remissions due to natural healing of the diseased subarachnoid tissues and cauda equina. Palliation is achieved to some degree by the use of pain-relieving drugs, by decreasing mobility, and by increasingly long periods of sitting or lying flat. Wheelchairs, electric wheelchairs and tricycles are often needed. For those under the age of 65 there is the prospect of financial help via the Motability Scheme of the Ministry of Transport. Disabled Car Badges are easily obtained. Every patient travels the long pilgrimage to various pain clinics, and must find for himself which drugs give most relief. Treatments which have been tried and found of little benefit are physiotherapy, transcutaneous nerve stimulation, dissection of scar tissue from the cauda equina, and injection of corticosteroids. Treatment with intrathecal hyaluronidase has been recommended[17], but the results have not been confirmed.

There is evidence that some patients benefit from insertion of micro-electrodes into the mid-dorsal spinal column, with electrical stimulation from a subcutaneous implanted battery or radio-receiver[18]. The treatment is costly, but worthwhile for the 60% subjective improvement of pain, 40% substantial reduction of medication and 26% increase in working capacity over a 4-year follow-up period.

Conclusion

The relentless and progressive pain syndrome of arachnoiditis is taxing to the patient's morale. In many instances doctors, relatives, and friends fail to realize that the pain can be as bad as terminal cancer, without the prospect of death to end the suffering. Well-meaning enquiries as to whether there is any improvement with the implication that there must inevitably be improvement, 'since it is not cancer', are distressing to the patient. There are sympathetic doctors, relatives, and friends who expect the patient to be brave, stoical, and cheerful. In the end the patient yearns for less exhortation and more compassion. Compassion is an important consequence of comprehension of the existence and nature of arachnoiditis.

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